Anesthesiology
55:409–415, 1981

Effect of Positive End-Expiratory Pressure on Left Ventricular Mechanics in Patients with Hypoxemic Respiratory Failure

R. M. Prewitt, M.D., L. Oppenheimer, M.D., J. B. Sutherland, M.D., L. D. H. Wood, M.D.†*

When positive end-expiratory pressure (PEEP) is added to intermittent positive pressure ventilation, cardiac output and stroke volume frequently fall despite unchanged or increased transmural left ventricular end-diastolic pressure. To determine whether a part of the fall in stroke volume with PEEP is explained by depressed left ventricular systolic function (increased end-systolic volume at a given end-systolic pressure on PEEP) the authors measured left ventricular end-diastolic volume (EDV), end-systolic volume (ESV), and the corresponding pressures in nine patients with acute hypoxemic respiratory failure. Measurements were made before and after 10 cm H_2O PEEP was added to the ventilator. PEEP reduced mean stroke volume from 71 to 62 ml and this was explained entirely by a reduction in end-diastolic volume from 135 to 112 ml ($P < 0.005$). Despite reduced EDV, pulmonary wedge pressure increased from 12 to 14 torr on PEEP, indicating reduced diastolic compliance or unstressed volume of the left ventricle in these patients similar to that reported in dogs. The authors conclude that PEEP reduces venous return and cardiac output without depressing left ventricular pumping function because end-systolic volume decreased from 64 to 49 ml on PEEP despite identical blood pressures (78 torr). They speculate that PEEP might improve ventricular performance by increasing intrathoracic pressure and left ventricular pressure relative to systemic blood pressure in extrathoracic vessels. (Key words: Heart: cardiac output; myocardial function; ventricles. Ventilation: positive end-expiratory pressure.)

IN PATIENTS with hypoxemic respiratory failure due to large intrapulmonary shunts ($\dot{Q}_s/\dot{Q}_t$), pulmonary oxygen exchange often improves dramatically when positive end-expiratory pressure (PEEP) is employed.[1,2] However, cardiac output (CO) often decreases unacceptibly on PEEP.[1,2] This is due in part to the reduced gradient for venous return which occurs when right atrial pressure rises with pleural pressure (P_{pl}) on PEEP.[3,4] By this mechanism, end-diastolic volume (EDV) and transmural (T_m) end-diastolic pressure (EDP-P_{pl}) of both ventricles decrease on PEEP, and return to normal on expansion of the central blood volume.[5] Yet recent investigations of canine models of hypoxemic respiratory failure demonstrated that transmural ventricular pressures did not decrease when stroke volume decreased on PEEP.[6-9] Furthermore, to maintain stroke volume and stroke work on PEEP required greater transmural left ventricular pressures than before PEEP.[6,8] One interpretation of these data is that PEEP depressed left ventricular systolic pumping function, accounting for part of the reduction in stroke volume and cardiac output.

That PEEP adversely affects left ventricular contractility in otherwise very ill patients is a worrisome possibility which might not be true if the effect of PEEP were in fact to increase the diastolic filling pressure of the left ventricle at each diastolic volume. The purpose of this study was to measure left ventricular end-diastolic and end-systolic volume-pressure (V-P) relationships to determine whether PEEP depressed left ventricular systolic pumping function in patients with hypoxemic respiratory failure. To illustrate our rationale, we calculated and present in figure 1 the two possible V-P relationships accounting for the data in our previous canine study of PEEP.[8] Transmural LVEDP was increased from 7 torr on zero end-expiratory pressure (Z_l) to 17 torr to maintain the same stroke volume on PEEP (P_2). To the extent that the diastolic V-P curve does not change with PEEP, LVEDV must increase with LVEDP on PEEP as indicated in figure 1*A*. Since stroke volume and blood pressure did not change between Z_1 and P_2,

ABBREVIATIONS

$B\bar{p}$	= mean arterial pressure
CO	= cardiac output
EDP	= end-diastolic pressure
EDV	= end-diastolic volume
ESP	= end-systolic pressure
ESV	= end-systolic volume
EF	= ejection fraction
LVEDP	= left ventricular end-diastolic pressure
MDS	= Medical Data Systems
MUGA®	= Multigated acquisition cardiac analyses
PCWP	= pulmonary capillary wedge pressure
PEEP	= positive end-expiratory pressure
P_{pl}	= pleural pressure
P_o	= pressure outside the heart
$\dot{Q}_s/\dot{Q}_t$	= right to left shunt
RVEDP	= right ventricular end-diastolic pressure
SV	= stroke volume
T_m	= transmural
ZEEP	= zero end-expiratory pressure

* Fellow of the Canadian Heart Foundation.

† Scholar of the Canadian Life Insurance Association.

Received from the Departments of Nuclear Medicine, Medicine and Surgery, University of Manitoba, Faculty of Medicine, Health Sciences Centre, Winnipeg, Manitoba Canada. Accepted for publication March 28, 1981. Supported by the Canadian Heart Foundation and the Medical Research Council of Canada. Presented at the annual meeting of the American Thoracic Society, Las Vegas, Nevada, 1979.

Address reprint requests to Dr. L. D. H. Wood: F2, Respiratory Lab, Health Sciences Centre, 700 William Avenue, Winnipeg, Manitoba, R3E 0Z3 Canada.

0003-3022/81/1000/0409 $00.85

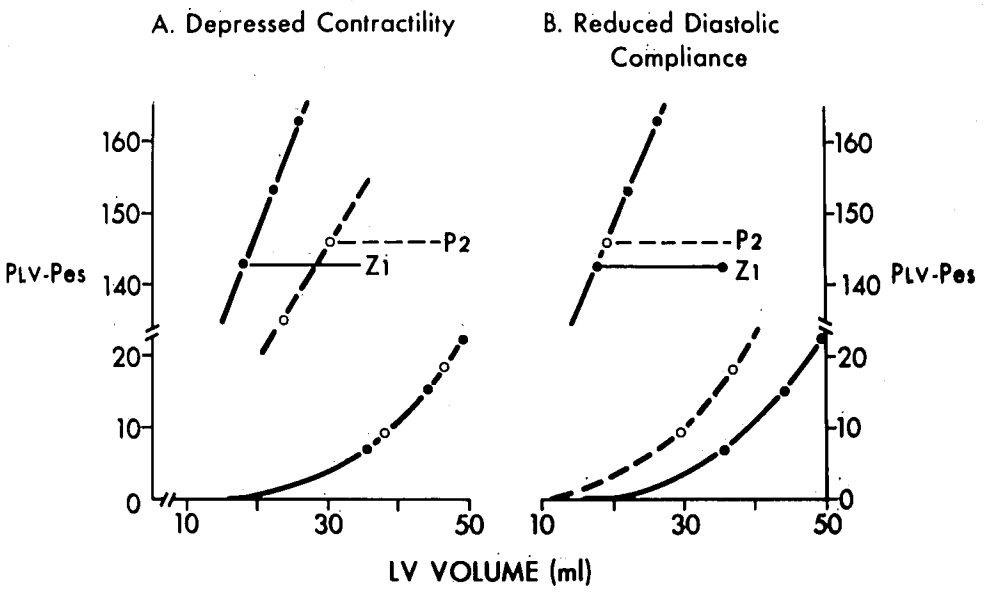

FIG. 1. Ordinates; transmural left ventricular pressure: Abscissae; left ventricular volume. Closed circles denote mean values off PEEP and open circles denote mean values on PEEP from reference 8. Stroke volume in condition P_2 is the same as in Z_1, yet the filling pressure is 10 torr higher at P_2. This might be due to depressed left ventricular pumping function on PEEP (interrupted systolic V-P relationship in panel A), or to reduced diastolic compliance or unstressed volume on PEEP (interrupted line, panel B).

end-systolic volume (ESV = EDV − SV) increased at the same left ventricular end-systolic pressure (LVESP). According to Sagawa *et al.*,[10,11] the rightward displacement of the end-systolic V-P curve indicates depressed left ventricular pumping function on PEEP, in that the ventricular muscle did not shorten as far against the same end-systolic pressure. Conversely, if PEEP does not depress left ventricular pumping function, end-systolic volume will not be increased at the same blood pressure on PEEP, as indicated in figure 1*B*. Then LVESV and stroke volume are the same in conditions Z_1 and P_2, so end-diastolic volume must also be the same despite the greater transmural LVEDP on PEEP. This explanation requires a reduction in the diastolic compliance or in the unstressed volume of the left ventricle on Peep as depicted in figure 1*B*. Note that if the only effect of PEEP were to impede venous return by raising right atrial pressure an amount equal to the increase in P_{pl}, reduced right ventricular output and reduced LVEDV would be associated with a reduction in transmural LVEDP along a diastolic V-P curve unaffected by PEEP. Then the ventricle would eject to the same end-systolic volume during PEEP as it did before PEEP, so reduced stroke volume on PEEP would be due to reduced LVEDV and PCWP with no change in left ventricular mechanics.

Methods

Nine patients were studied in the Intensive Care Unit and Surgical Recovery Room at the Health Sciences Centre (table 1). The age range was from 23–79 years. All patients were being ventilated for acute, hypoxemic respiratory failure of various etiologies, and their pulmonary shunt exceeded 20 per cent on zero end-expiratory pressure during oxygen ventilation. None had evidence of acute myocardial injury at the time of the study, and all had adequate peripheral circulation by clinical examination. Patients were receiving constant infusions of the following drugs ($\mu g \cdot kg^{-1} \cdot min^{-1}$) at the time of study: Patient 4, nitroprusside, 0.8; Patient 5, dopamine, 1.5; Patient 6, dopamine, 2.0, nitroprusside, 0.6; Patient 7, dopamine, 4.0, nitroprusside, 0.4. Patients did not receive diuretics or significant volume infusion during the period of study. Institutional review and approval of the protocol was required and was obtained. Informed consent concerning the nature and purpose of the study was obtained in each case from the patient or from the nearest relative.

The patients were monitored with thermodilution Swan-Ganz catheters from which measurements of CO and PCWP were obtained. This catheter was connected to a pressure transducer (Bell & Howell, 4-327I or Statham® P23 BB), the output of which was displayed on a Space Labs Physiological monitor. Cold 5-ml boluses (5 per cent D/W) were injected in the proximal lumen of the Swan-Ganz catheter for CO determinations and the thermal dilution curve was analyzed by computer (Columbus Instruments). The accuracy and reproducibility of the cardiac output estimates was verified in a bench model of pulsatile blood flow. Mixed venous blood was sampled and analyzed for gas tensions (Corning® 165-2) and hemoglobin concentrations. A second catheter had been placed via the femoral artery in the aorta to obtain samples of arterial blood for similar analysis of blood-gas tensions, and to continuously monitor systemic blood pressure via connection to a Bell & Howell® pressure transducer and display on a Space Labs monitor. Oxygen saturation was estimated from blood-gas ten-

TABLE 1. Clinical Data

Patient Number	Age (yr)	Sex	Diagnosis	Pulmonary Complications	Right-to-Left Shunt (Per Cent Cardiac Output) (At time of Study, no PEEP)
1	53	M	Trauma, peritonitis; sepsis. No evidence of previous cardio-pulmonary disease.	Low pressure pulmonary edema	28
2	51	M	Hemorrhagic pancreatitis; acute renal failure; septicemia. No history of prior cardio-pulmonary disease.	Low pressure pulmonary edema.	27
3	23	F	Diabetes mellitus; diabetic nephropathy; cadaver transplant; staphylococcal septicemia; No history of previous cardiopulmonary disease.	Pneumocystis pneumonia; cytomegalovirus pneumonia	36
4	73	M	Alcoholic, chronic bronchitis, subarachnoid hemorrhage, complicated by coma	Pneumonia and atelectasis	22
5	56	F	Chronic lymphocytic leukemia, herpes zoster encephalitis; Past history of bronchitis; No previous cardiac history	Pneumonitis (probably varicella) bacterial pneumonia	21
6	76	F	Chest and abdominal trauma; bacteremia; fluid overload. No previous history of pulmonary disease. EKG: old anterior MI.	Pulmonary contusion, fluid overload, pulmonary edema.	25
7	79	M	Pancreatitis; G.I. bleed, acute renal failure, thrombocytopenia with hemorrhage; volume overload, sepsis	Volume overload pulmonary edema; possible low pressure pulmonary edema	35
8	40	M	Graves disease; coma; thyroid storm, seizures; pulmonary embolus; No prior history of cardiopulmonary disease.	Pneumonia, lung abscess	25
9	53	M	Pancreatitis; acute renal failure, bleeding diathesis; Gram-negative septicemia with shock; No history of prior cardiopulmonary disease.	Low pressure pulmonary edema	24

sions, and mixed venous ($C\bar{v}_{O_2}$) and arterial (Ca_{O_2}) oxygen contents were calculated. To avoid hypoxemia, the patients were ventilated with oxygen during each study, and arterial blood O_2 saturation was always complete. Samples of mixed expired gas were analyzed for PE_{O_2} and PE_{CO_2} to estimate ideal alveolar P_{O_2} ($PA_{O_2} = PE_{O_2} + PE_{CO_2} - Pa_{CO_2}$, and $\dot{Q}_s/\dot{Q}_t$ was calculated as .003 $(PA_{O_2} - Pa_{O_2})/(Cc'_{O_2} - C\bar{v}_{O_2})$.

Left ventricular ejection fraction (EF) was measured using an established equilibrium nuclear cardiology technique.[12,13] Patients' red cells were labeled using an *in vivo* technique. Ten milligrams of the reducing agent stannous pyrophosphate was injected intravenously and this bound to the reduced red blood cells. After equilibration of the labeled cells, a mobile gamma camera (Picker®), equipped with a parallel hole collimator, was oriented in a modified left anterior oblique position.[12] The camera head was positioned so that the interventricular septum was clearly visible. Left ventricular imaging was accomplished by storing counts and the EKG QRS complex on magnetic tape, contained in the mobile camera. Recording of three to four hundred cardiac cycles gave sufficient counts to produce high resolution images at specific points in the cardiac cycle. Recording time for a single EF determination was 3–5 min. Following completion of the entire study, the information stored on tape was transferred to computer (MDS) for further analyses.

The MDS computer uses the R wave of the QRS complex as a gating signal and divides the R-R interval of each of the 300 to 400 cardiac cycles into 28 equal

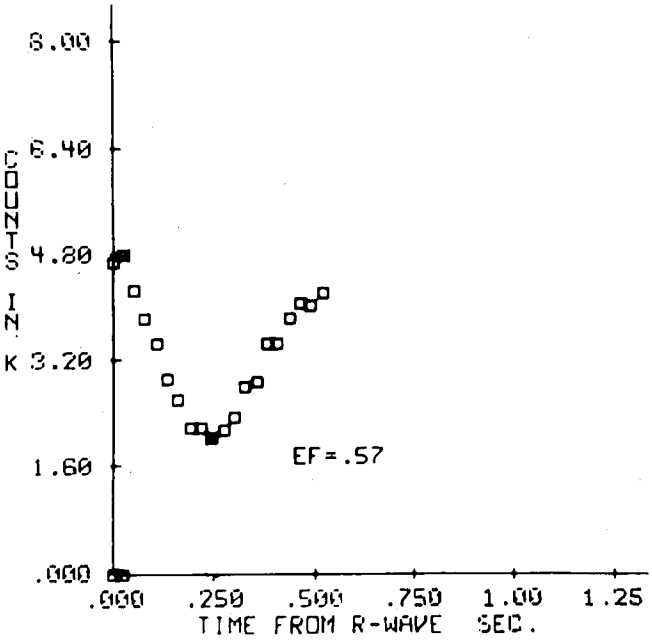

FIG. 2. Computer-generated time-activity curve during one composite cardiac cycle divided into 28 equal time segments (actually the integration of approximately 500 cardiac cycles, see text). The beginning of the curve coincides with the R-wave. The background corrected EF (0.57) is automatically displayed.

time segments (MUGA® analysis). Each of the segments has a corresponding storage area and a high count image, the sum of all cycles, is accumulated for each segment. Following data acquisition a second MDS program (MUGA), using the second derivative technique,[12] automatically detects the edge of the left ventricle and generates a background corrected, time-activity curve (fig. 2). From the curve the computer automatically calculates the left ventricular EF. Ejection fraction was determined two to four times for each experimental condition. The computer EF did not vary by more than 0.02 and mean values are reported. We verified our use of this technique using a pulsating balloon having a measured stroke volume of 18–48 ml and an end-ejection volume of either 70 or 120 ml.

While the gated cardiac study was being acquired, repeated measurements of CO, HR, PCWP, and Bp were obtained. Between 6 and 12 determinations of each variable were recorded during each condition, and the mean for that period was determined. If the patient was not already on PEEP when the study began, the above measurements were first obtained during ventilation with zero end-expiratory pressure (ZEEP), then 5 min after the addition of 10 cm H_2O PEEP, and finally to control for time, 5 min after removal of Peep. The entire study took approximately 1 hour to complete. The mean of the measurements obtained during ventilation with ZEEP was compared to the corresponding values obtained during ventilation with PEEP. If the patient was on PEEP at the time of the study then measurements on PEEP bracketed those on ZEEP. SV was determined by dividing CO by HR, and left ventricular EDV = SV/EF and ESV = EDV − SV. The measured and calculated values were compared between PEEP and ZEEP by Student's paired t test.

Results

Table 2 presents individual and mean hemodynamic measurements. Cardiac output determinations were quite reproducible in each patient and in each condition studied, and the coefficient of variation was always less than 15 per cent. Cardiac output decreased on PEEP in eight patients and increased in one patient. Mean CO decreased from 7.0 ± 2.6 on ZEEP to 6.3 ± 2.1 l/min on PEEP ($0.05 < P < 0.1$).

Stroke volume decreased on PEEP in all but Patient 2, and mean SV decreased from 71 ± 17 to 62 ± 16 Ml ($P < 0.05$). Ejection fraction on ZEEP ranged from 0.24 to 0.72 (mean 0.56 ± 0.15) and it increased on PEEP in six of nine patients to a mean value of 0.59 ± 0.14. Accordingly, LVEDV was reduced on PEEP in eight of nine patients, and the mean LVEDV decreased by 23 ml from 135 ± 41 on ZEEP to 112 ± 41 on PEEP ($P < 0.005$). Despite reduced LVEDV on PEEP, PCWP increased on PEEP in eight of nine patients, and mean PCWP increased from 12 (ZEEP) to 14 torr (PEEP).

Left ventricular end-systolic volume decreased on PEEP in all patients, and mean LVESV decreased from 64 ± 44 on ZEEP to 49 ± 36 ml on PEEP ($P < 0.005$). The reduced LVESV on PEEP was not associated with reduced blood pressure which was similar in all patients (78 ± 19 torr on ZEEP; 77 ± 16 torr PEEP).

Left ventricular volume-pressure coordinates for all nine patients are shown in figure 3. In each patient, either two ZEEP or two PEEP coordinates are displayed and numbered to indicate the value before (1) and after (2) the intervention. The diastolic coordinates on PEEP in patients 1 and 3–9 all show reduced LVEDV for a similar PCWP. The systolic volume-pressure coordinates in patients 2–7 demonstrate reduced LVESV for a similar Bp, and in patients 1, 8, and 9 they are compatible with that possibility. These results are not dependent on whether the study began with ZEEP or PEEP. Returning to the first condition after the intervention produced a randomly varied response in terms of the four variables measured, and the mean of the first and second measurements are presented in table 2.

Discussion

The effects of PEEP on left ventricular mechanics in these patients may be summarized as follows. Mean

TABLE 2. The Effect of PEEP on Left Ventricular Mechanics in Patients with Hypoxemic Respiratory Failure

Patient Number	CO (l/min)		SV (ml)		PCWP (torr)		LVEDV (ml)		$\bar{Bp}$ (torr)		LVESV (ml)		EF	
	Z	P	Z	P	Z	P	Z	P	Z	P	Z	P	Z	P
1	6.9 ± 0.1	5.8 ± 0.9	62	52	9	12	98	77	105	99	40	25	0.65	0.68
2	6.5 ± 0.4	7.8 ± 0.5	63	73	8	11	110	111	46	50	47	41	0.57	0.64
3	7.1 ± 0.5	6.2 ± 0.6	78	56	7	8	108	79	72	71	30	23	0.72	0.71
4	5.0 ± 0.3	4.4 ± 0.1	64	46	13	16	92	62	87	87	28	16	0.70	0.74
5	8.6 ± .82	6.4 ± 0.5	65	48	8	10	129	98	62	62	65	48	0.50	0.49
6	5.7 ± 0.6	5.5 ± 0.4	61	58	25	21	116	95	84	84	51	37	0.55	0.61
7	5.5 ± 0.4	5.3 ± 0.2	54	52	16	18	225	185	80	84	171	133	0.24	0.29
8	13.1 ± 6.2	11.1 ± 0.9	110	95	8	11	162	152	100	94	52	47	0.68	0.67
9	4.3 ± 0.3	4.1 ± 0.5	83	77	12	15	173	156	64	63	90	79	0.48	0.49
MEAN	7.0 ± 2.6	6.3 ± 2.1	71 ± 17	62* ± 16	12 ± 6	14 ± 4	135 ± 41	112* ± 41	78 ± 19	77 ± 16	64 ± 44	49* ± 36	0.56 ± 0.15	0.59 ± 0.14

Values are means ± SD.
For description of symbols see abbreviations.

* Denotes difference ($P < 0.05$) from the corresponding value.

stroke volume decreased by 9 ml on PEEP. This was entirely attributable to the change in mean left ventricular end-diastolic volume, which decreased by 23 ml despite a small increase in PCWP. Mean left ventricular end-systolic volume also decreased significantly on PEEP despite similar aortic pressure. These features are illustrated in figure 4, which shows the mean diastolic and systolic volume-pressure coordinates for ZEEP (open circles) and PEEP (closed circles). The solid lines are hypothetical diastolic and systolic volume-pressure curves drawn through the ZEEP coordinates to approximate the slope and position of such curves from the literature.[10,11]

Because LVEDV decreased on PEEP, yet PCWP increased, the corresponding diastolic V-P curve during PEEP must be shifted to the left, as indicated by the interrupted diastolic curve in figure 4. Such a reduction in diastolic compliance or unstressed volume of the left ventricle on PEEP was recently described in three independent canine studies,[14-16] so our data extend their results to patients for the first time. Note, however, that we did not measure the change with PEEP in pressure outside the heart (ΔP_o), so the diastolic closed circle in figure 4 is not true transmural pressure unless ΔP_o was zero. Both theory and canine measurements predict that the change in pleural pressure is about half the added PEEP.[5-9] Conceivably, constraint of the diastolic myocardium by the distended lung or by a pericardium stretched by its attachments to a low diaphragm on PEEP could make $\Delta P_o \gg \Delta P_{pl}$. Indeed, the plotted open square in figure 4 indicates the lowest possible transmural pressure during PEEP, calculated for the condition ΔP_o = PEEP. This transmural pressure falls on the ZEEP diastolic curve, indicating that LVEDV and the transmural LVEDP decrease on PEEP along the unchanged diastolic volume-pressure characteristics of the myocardium. In other words, PEEP reduced stroke volume in these patients by reducing venous return, right ventricular output, and LVEDV, but PCWP did not decrease because the pressure outside the left ventricle increased on PEEP.

Despite reduced stroke volume and stroke work at increased PCWP on PEEP, left ventricular systolic function was not depressed. This conclusion is based on our observation that LVESV decreased PEEP in all patients at the same aortic pressure, so the end-systolic volume-pressure relationship on PEEP must lie significantly to the left of the ZEEP curve as indicated by the interrupted systolic line in figure 4. Rather than indicating depressed LV pumping function, such a result indicates improved ventricular function on PEEP in that the left ventricle shortened farther against the same aortic pressure.[10,11] Indeed, had LVESV not decreased on PEEP, mean stroke volume would decrease by 23 ml (equal to the reduction in LVEDV) instead of the 9-ml reduction observed.

We know no mechanism by which PEEP might improve ventricular pumping function unless reduced LVESV is due to reduced transmural end-systolic pressure. For example, when ΔP_o = PEEP, the transmural aortic pressure lies on the ZEEP systolic V-P curve as illustrated by the systolic square in figure 4. In effect, increased pressure outside the heart on PEEP raises ventricular systolic pressure relative to extrathoracic vessels, allowing ejection to proceed to a lower ventricular volume. Note that the systolic effect of PEEP we describe is similar to that noted following the administration of nitroprusside or nitroglycerin. If myocardial ischemia exists, PEEP will improve ventricular pumping function by diminishing cavitary volume to relieve ischemia. Although our patients had no clinical or EKG evidence of coronary insufficiency, such global ischemia in otherwise ill patients might go undetected. However, their cardiac output and peripheral circulation seemed adequate at relatively normal PCWP on ZEEP, so we think it unlikely that PEEP reduced ischemia. We conclude that

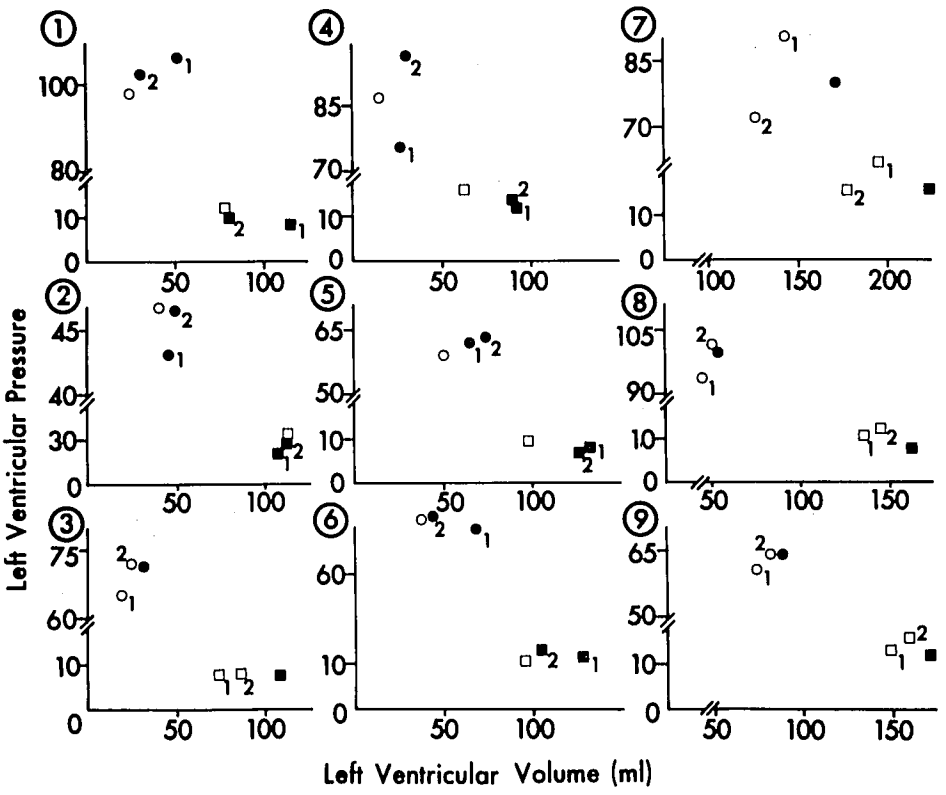

FIG. 3. Left ventricular end-diastolic (squares) and end-systolic (circles) volume-pressure relationships on PEEP (open symbols) and off PEEP (closed symbols) in nine patients. Ordinates: Mean aortic pressure (systolic points) and mean PCWP (diastolic points). Abscissae: Left ventricular end-systolic and end-diastolic volumes. The subscripts (1 or 2) indicate whether the volume-pressure coordinates during a given ventilatory condition (ZEEP or PEEP) were obtained before (1) or after (2) the alternative ventilatory condition. The circled numbers at the upper left-hand corner of each graph indicate the case number. Note that PEEP reduced both end-diastolic and end-systolic volume in most cases without reducing the corresponding pressures.

10 cm H_2O PEEP did not depress left ventricular systolic function in these patients, and point out that this result agrees with recent observations in canine oleic acid edema.[14]

Our results and conclusions rely on reproducible measurements of ejection fraction and stroke volume to estimate changes in left ventricular end-diastolic and end-systolic volumes with PEEP, and rely on PCWP and BP to estimate corresponding changes with PEEP in LVEDP and LVESP, respectively. Excellent correlation has been obtained between left ventricular ejection fractions determined from contrast angiography and gated radionuclide studies using the same methods as in this study.[12,13] The good reproducibility of thermodilution cardiac output determination in severe lung disease was confirmed in this study (note individual standard deviation, table 2). Results from this technique correlate well with Fick and dye dilution estimates of cardiac output. We contend that inaccuracies in cardiac output or ejection fraction are unlikely to be introduced by PEEP, so the systematic and reversible reduction of LVEDV and LVESV on PEEP are real. The ventricular pressure at which the aortic valve closes is best signalled by the onset of the dicrotic notch in a high-fidelity recording of aortic pressure. This is closely approximated in most clinical and experimental conditions by the mean aortic blood pressure, and this pressure of end ejection has been used to indicate end-systolic pressure.[10,11] PCWP is an indirect but less invasive estimate of left ventricular diastolic pressure having several potential artifacts.[9] In this study, the pressure wave form was carefully examined during PCWP determinations on PEEP to avoid the overestimation which occurs when alveolar pressure exceeds left atrial pressure. All pressure waves had a normal wedge pattern, and the measured PCWP exceeded alveolar pressure on PEEP in all but Patient 3. In that patient, we confirmed that the catheter tip lay posterior to the left atrium on a lateral x-ray. Accordingly, all PCWP values reported were obtained in Zone III conditions, in which changes in PCWP accurately estimated changes in LVEDP.[9] Conceivably, in the presence of severe lung disease such accurate estimates of PCWP exceed left atrial pressure due to pulmonary venous resistance, and this overestimate changes with PEEP. In a canine model of hypoxemic respiratory failure[9] this did not occur until high levels of PEEP (>15 cm H_2O). We conclude that our estimates of LV volumes and pressures accurately measure the changes which occur with PEEP.

Accordingly, 10 cm H_2O PEEP reduces cardiac output and stroke volume by lowering venous return, right ventricular output, and LVEDV in patients with acute hypoxemic respiratory failure. PEEP does not reduce left ventricular pumping function in these patients. Rather, the pulmonary wedge pressure fails to decrease when LVEDV and stroke volume decrease on PEEP because the left ventricular filling is restricted by the surrounding

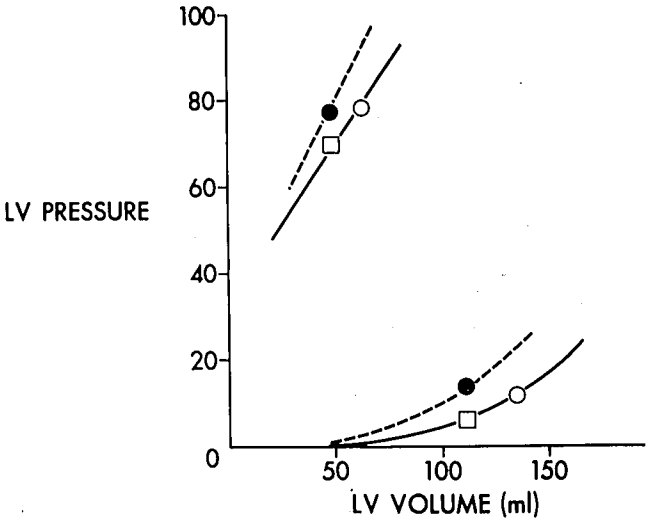

FIG. 4. Effect of PEEP on diastolic and systolic volume (abscissa)-pressure (ordinate) relationships of the left ventricle in patients with hypoxemic respiratory failure. Open circles denote the mean (n = 9) volume-pressure coordinates on ZEEP, and the continuous lines are drawn through these points to indicate typical volume-pressure curves. Closed circles denote the mean (n = 9) volume-pressure coordinates on PEEP. Both LVESV and LVEDV are significantly reduced at the same pressure, indicating a leftward displacement of the volume-pressure curves (interrupted lines). Note that the change in pressure outside the heart (ΔP_o) with PEEP was not measured, and these curves are true if $\Delta P_o = 0$. If ΔP_o = PEEP, the mean transmural pressures on PEEP are denoted by (□), which fall on the ZEEP curves. Accordingly, these data do not clearly demonstrate that PEEP altered either the diastolic or the systolic volume-pressure curves. They do exclude the possibility that PEEP depressed ventricular pumping function.

lung or pericardium. Although those patients whose cardiac outputs drop on PEEP are relatively hypovolemic despite increased PCWP, we do not infer that plasma volume expansion is indicated, for that therapy increases PCWP further. Since increased PCWP increases low pressure edema,[17] we advocate less PEEP or vasoactive agents[18] to restore cardiac output.

The authors thank G. W. Dean, Ph.D., and R. Palser, Ph.D. of the Department of Nuclear Medicine, who assisted with the validation and analysis of ejection fraction.

References

1. Ashbaugh DG, Petty TL, Bigelow DB, et al: Continuous positive pressure breathing (CPPB) in adult respiratory distress syndrome. J Thorac Cardiovasc Surg 57:31–41, 1969
2. Pontoppidan H, Wilson RS, Rie MA, et al: Respiratory intensive care. ANESTHESIOLOGY 47:96–116, 1977
3. Cournand A, Motley HL, Werko L, et al: Physiological studies of the effects of intermittent positive pressure breathing on cardiac output in man. Am J Physiol 162:174–180, 1947
4. Fermoso JD, Richardson TQ, Guyton AC: Mechanism of decrease in cardiac output caused by opening the chest. Am J Physiol 207:1112–1116, 1964
5. Qvist J, Pontoppidan H, Wilson RS, et al: Hemodynamic responses to mechanical ventilation with PEEP ANESTHESIOLOGY 42:45–55, 1975
6. Scharf SM, Caldini P, Ingram RH: Cardiovascular effects of increasing airway pressure in the dog. Am J Physiol 232:H35–H43, 1977
7. Scharf SM, Ingram RH: Effects of decreasing lung compliance with oleic acid on the cardiovascular response to Peep. Am J Physiol 233:H635–H641, 1977
8. Prewitt RM, Wood LDH: The effect of positive end-expiratory pressure on ventricular function in dogs. Am J Physiol 5:H534–H544, 1979
9. Cassidy SS, Robertson CH, Pierce AK, et al: Cardiovascular effects of positive end-expiratory pressure in dogs. J Appl Physiol 44:743–750, 1978
10. Sagawa K: the ventricular pressure-volume diagram revisited. Circ Res 43:677–687, 1978
11. Sagawa K, Suga H, Shoukas AA, et al: End systolic pressure/volume ratio: A new index of ventricular contractility. Am J Cardiol 40:748–753, 1977
12. Ashburn WL, Schelbert HR, Verla JW: Left ventricular ejection fraction—A review of several radionuclide angiographic approaches using the scintillation camera. Prog Cardiovasc Dis 20:267–284, 1978
13. Burrow R, Strauss HW, Singleton R, et al: Analysis of left ventricular function from multiple gated acquisition (MUGA) cardiac blood pool imaging: Comparison to contrast angiography. Circulation 56:1024–1031, 1977
14. Wood LDH, Prewitt RM. Cardiovascular management in acute hypoxemic respiratory failure. Am J Cardiol 47:963–972, 1981
15. Fewell JE, Abendschein DR, Carlson CJ, et al. Continuous positive pressure ventilation decreases right and left ventricular end-diastolic volume in the dog. Circ Res 46:125–132, 1980
16. Haynes JB, Carson SD, Whitney WP, et al: Positive end-expiratory pressure shifts left ventricular diastolic pressure-area curves. J Appl Physiol 48:670–676, 1980
17. Prewitt RM, McCarthy J, Wood LDH: Treatment of acute low pressure pulmonary edema in dogs. J Clin Invest 67:409–418, 1981
18. Hemmer M, Suter PM: Treatment of cardiac and renal effects of Peep with Dopamine in patients with acute respiratory failure. ANESTHESIOLOGY 50:399–403, 1979